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METABOLIC AND ENDOCRINE CHANGES IN HEPATIC SCHISTOSOMIASIS.(U)  
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METABOLIC AND ENDOCRINE CHANGES IN  
HEPATIC SCHISTOSOMIASIS

FINAL SCIENTIFIC REPORT

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Albumin was decreased in hepatic schistosomiasis while the globulins were increased. Serum amino acids showed significant decrease of glutamine, glutamic acid and valine. Severe impairment of ammonia tolerance correlated with increase transaminases levels.

Total serum thyroxine, triiodothyronine uptake and free thyroxine index were comparable to controls, follicle-stimulating and luteinizing hormones significantly higher, urinary estrogens increased and urinary 17 - ketosteroids decreased.

The prothrombin and activated partial thromboplastin times were prolonged, thrombocytopenia was present, platelet adhesiveness decreased, platelet aggregation delayed, fibrinogen content was normal and fibrinolytic activity accelerated in schistosomiasis.

Histochemistry of the liver revealed enzymatic abnormalities in spite of mild impairments of liver functions.

Metabolic and Endocrine Changes  
in Hepatic Schistosomiasis

M.H.Ghanem, Mofid H.Fahmy and M. Said

ABSTRACT

In hepatic schistosomiasis the glucose disappearance rate was slower than in controls, plasma insulin levels were comparable to that of controls up to 60 minutes after glucose loading and higher at 90 minutes, growth hormone levels<sup>were</sup> comparable to controls and free fatty acids significantly higher.

Total lipids, cholesterol, phospholipids, triglycerides and alpha-lipoproteins were significantly lower than in controls. The mean total lipids, cholesterol and phospholipids were significantly lower in patients with collaterals than in patients without, the difference disappeared after decongestion operation. Fat tolerance tests showed less triglyceride increment in schistosomiasis and greater rise of free fatty acids with rapid elimination. The lipoprotein lipase activity was decreased and the total phospholipid value and their fractions differed from that of controls.

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Metabolic and Endocrine Changes in  
Hepatic Schistosomiasis  
Final Scientific Report

Schistosomiasis induces a special type of cirrhosis characterized by periportal fibrosis with minimal parenchymal damage and absence of regeneration.

In the present study patients with hepatic schistosomiasis were compared to normal controls. The diagnosis of hepatic schistosomiasis <sup>w</sup>as based on the clinical findings of an enlarged liver and spleen, the presence of intestinal or urinary schistosomiasis and confirmed by percutaneous liver biopsy. Cases where the schistosomal lesion was associated with other etiological factors were excluded. The liver involvement was uncomplicated with ascites or liver failure. Malnutrition, parasitic infestations and bacterial infections were controlled before investigation. All patients and controls were put on adequate well balanced diet for at least one month before investigation. Assessment of liver functions were made and none of the patients had jaundice or marked impairment of liver function tests. All patients and controls were males aged 18 to 51 years. In the present work we aimed at studying some aspects of carbohydrate, fat and protein metabolism in addition to some endocrinological studies in hepatosplenomegalic schistosomal patients. Great care was taken in the

selection of the patients so as to limit our study on the effect of schistosomal affection of the liver on these parameters. In most of the previous studies on hepatic schistosomiasis, other complicating factors were not excluded.

Glucose Tolerance, plasma insulin, growth hormone and free fatty acids:

Intravenous glucose tolerance tests (50ml of 50 percent glucose intravenously) were performed in forty-nine male hepatic schistosomiasis patients and twelve male controls. The fasting blood sugar levels were higher in schistosomal patients than in controls and the glucose disappearance rate was significantly slower (Table 1). The kg value was lower than 1 in 18 percent of the patients.

The maximum drop in blood sugar levels after exogenous insulin administration (0.1 unit/kg body weight) occurred at 30 minutes in both controls and hepatic schistosomiasis patients, the percent maximum drop was comparable in both groups (Table 2).

Determination of immunoreactive insulin (IRI) during intravenous glucose tolerance tests revealed a rise of plasma IRI within 4 minutes which reached maximum levels at 7 minutes in both controls and hepatic schistosomiasis patients. The

IRI levels were comparable in both groups except 90 minutes after glucose administration when plasma IRI was significantly higher in schistosomal patients (Table 3).

The plasma growth hormone levels (HGH) after insulin administration began to rise at 20 and 30 minutes, reached a maximum at 60 minutes and at 120 minutes was still much higher than the fasting level. There was no significant difference between controls and schistosomal patients (Table 4).

The fasting free fatty acids levels were significantly higher in hepatic schistosomiasis patients than in controls (Table 5).

Thus, glucose intolerance was demonstrated in hepatic schistosomiasis similar to other types of cirrhosis. The observed abnormality of carbohydrate metabolism is not related to malnutrition, gross obesity, underlying pancreatic insufficiency, corticosteroid or diuretic therapy, hypokalemia or old age, as all these complicating factors were avoided. Other possible etiologies for the glucose intolerance of liver disease include insulin resistance, impairment of glycogen storage and release, high plasma free fatty acids, impaired hepatic glucose uptake and decreased hepatic destruction of hormonal and non-hormonal factors that oppose the action of insulin.

The insulin levels that were comparable to that of controls in the fasting state and up to 60 minutes after glucose administration were associated with blood glucose levels above control values. These patients could therefore be considered to have a relatively decreased insulin secretion. In addition, high plasma free fatty acids level was suggested to decrease glucose tolerance by decreasing the overall oxidation of glucose.

Exogenous insulin resistance could not be demonstrated in compensated hepatic schistosomiasis. The higher fasting blood sugar levels in schistosomal patients is against impaired hepatic uptake of glucose. Excess growth hormone reported in other types of cirrhosis and which can produce a decrease in glucose tolerance was not demonstrated in hepatic schistosomiasis.

#### Blood Lipids and fat Tolerance Tests:

Forty-one patients with compensated hepatic schistosomiasis and <sup>3</sup>eighty-one controls were investigated. Estimations were made on total lipids (TL), serum triglycerides (TG), total cholesterol (CH), cholesterol ester (CE), percent esterification of cholesterol, phospholipids (PL) and alpha-lipoprotein percent. Eighteen of the schistosomal patients (group A) gave

a history of frank hematemesis from esophageal varices with evident collaterals demonstrated by splenoportography or evidence of esophageal varices by barium swallow. Fourteen other schistosomal patients (Group B) had no history of hematemesis with no evidence of collaterals or esophageal varices with splenoportography or barium swallow. Splenectomy and decongestion operation was performed on seven patients of group A and four patients of group B and the investigations were repeated again one month after the operation.

In schistosomal patients TL, CH, PL, TG as well as alpha<sub>2</sub>-lipoprotein percent were significantly lower than in the controls (Table 6). On comparing the serum lipids values in patients with (Group A) and without (Group B) portosystemic collaterals, the mean TL, CH, CE and PL were significantly less in group A than in group B while the difference in percent esterification of cholesterol and alpha<sub>2</sub>-lipoprotein percent were not significant (Table 7). After decongestion operation in group A patients, TL, CH, CE and PL showed highly increased levels while the percent esterification of cholesterol, TG and alpha-lipoprotein percent were not significantly changed after operation (Table 8). The same operation did not produce any significant change in serum lipids in group B patients (Table 9). No significant

difference was present in serum lipid levels in both groups A and B after the decongestion operation (Table 10).

The present results showed that serum lipids were significantly lower in schistosomal patients than controls. One of the main factors contributing to this decrease in serum lipids was the presence of portosystemic collaterals with diversion of portal blood to the systemic circulation. This is evidenced by the lower serum levels of TL, CH, CE and PL in patients with evident collaterals (group A) than those without (group B) and by the significant rise in these lipids in group A after the decongestion operation while no significant rise occurred in group B. The lowering of alpha-lipoprotein percent was attributed to impaired lipid binding capacity to apoprotein A. It was suggested that low lecithin cholesterol acyltransferase activity in plasma may be responsible for lowering of the percent esterification of cholesterol in liver disease. Other contributing factors responsible for low blood lipid levels were the defective intestinal absorption, unsaturated fatty acids as the main source of fat in Egyptian peasant's diet and the disturbed estrogen/androgen ratio.

An intravenous fat tolerance test was performed in thirteen hepatic schistosomiasis patients and thirteen controls by the intravenous injection of intralipid (10% fat emulsion) in a dose of 0.39/kg body weight. Blood samples obtained

before and after the intravenous fat administration were analysed for triglycerides (TG) and free fatty acids (F.F.A.) The fasting levels of serum triglycerides were significantly lower than that of controls. The mean values were  $66 \pm 13.6$  mg/100 ml and  $82 \pm 21.7$  mg/100 ml respectively. Ten minutes after intralipid administration the increment TG mean value was significantly less in schistosomal patients (Table 11). In controls the peak of plasma FFA was noticed 50 minutes after Intralipid administration whereas in schistosomal patients it was significantly higher and occurred at 30 minutes (Table 12). The rapid removal of the circulating TG and greater rise of plasma F.F.A. in schistosomal patients was postulated to be due to the greater uptake of TG by the enlarged liver and spleen, reduced removal of the circulating lipase and increased plasma volume in hepatic schistosomiasis. Also as the fasting plasma TG concentration in schistosomal patients is lower than that of controls, then the rate of removal is expected to be greater in these patients as compared to controls. The rapid elimination of the circulating plasma FFA in schistosomal patients can be attributed to the striking disappearance of the injected TG in these patients and hence marked diminution of the substrate on which the hydrolysing enzyme lipase acts.

The lipoprotein lipase activity ( ug glycerol/ml plasma/H) was determined in twelve hepatic schistosomiasis patients and ten controls in the fasting state and after intramuscular heparin injection. The rise in lipoprotein lipase activity 10 minutes after heparin was significantly less than in controls and after 20 minutes it did not return to the fasting levels unlike controls.

The total serum phospholipids and the relative and absolute values of their fractions were studied in 77 male hepatic schistosomiasis patients and 200 healthy controls. Both controls and patients were subdivided into those 35 years of age and below and those above 35 years of age. The mean total phospholipid value in the schistosomal patients was significantly lower in the older age group and did not significantly increase with age unlike controls. The relative and absolute values of lysolecithins showed significant difference from that of controls in both age groups. This is due to liver dysfunction, increased acylation of lysolecithin to lecithin, increased lysolecithinase activity secondary to increased estrogens and decreased activity of lecithin cholesterol acyltransferase. The sphingomyelins and cephalins values showed a significant decrease in the mean absolute values in the older age group because of diminished synthesis and increased methylation of phosphatidylethanolamine to phosphatidylcholine.

While the relative value of lecithin was significantly increased in the older age group, yet its mean absolute value was decreased in the same age group. The relative increase of lecithins is due to increased acylation of lysolecithin to lecithin and reduced activity of lecithin cholesterol acyltransferase.

Serum proteins & serum and urinary amino acids:

Total serum proteins values in 49 hepatic schistosomiasis patients were higher than in controls, the increase being mainly due to marked rise of serum  $\gamma$ -globulin levels (Table 13). Serum albumin levels though were in the lower limit of normal yet they were significantly decreased while  $\alpha_1$ -globulins,  $\alpha_2$ -globulins,  $\beta$ -globulins and especially  $\gamma$ -globulins, were significantly increased.

In twenty-three hepatic schistosomiasis patients and eight controls, plasma amino acids were determined by paper chromatography and colorimetry. Most of the plasma amino acids levels in schistosomiasis were comparable to controls except glutamine, glutamic acid and valine which were significantly decreased and methionine which was increased (Table 14). The decrease in some plasma amino acids may be due to disturbed intermediary metabolism or increased amino acid excretion. Determination of urinary amino acid excretion in hepatic schistosomiasis patients and controls showed that the urinary excretion of glutamine, glutamic acid and valine were comparable to controls with no evidence of over-excretion (Table 15). The increased excretion of methionine is the result of its increased plasma levels. On

the other hand, while there was no significant difference between the plasma levels of cystine and cysteine, lysine, threonine, tyrosine, phenylalanine and tryptophane in schistosomel patients and controls, their urinary excretion was significantly increased in schistosomal patients. This may be due to impaired reabsorption in the renal tubular cells.

#### Ammonia Tolerance

Fasting blood ammonia levels were determined in seventeen patients with hepatosplenic schistosomiasis. An oral loading dose of ammonium chloride (5g) was administered and ammonia tolerance determined (Table 16) by calculating the increment (per cent increase above fasting levels) at 30, 60, 90 and 120 minutes. The patients investigated could be divided into two groups, group A composed of thirteen patients with mild impairment of ammonia tolerance and group B of four patients with severe impairment of tolerance. In group B the increment ammonia mean value was significantly higher at all times with only a partial return at 120 minutes. The peak of blood ammonia was noticed at 60 minutes in both groups.

On comparing the results of liver function tests in both groups (Table 17), a significant increase of transaminasis levels was detected in group B patients with severely impaired ammonia tolerance.

All patients had splenomegaly and abdominal collaterals could be detected in both groups. The degree of impairment of ammonia tolerance could not be correlated with size of spleen or degree of collaterals.

Endocrinological Studies:

Many endocrine changes were reported in patients who develop hepatosplenic schistosomiasis. A state of "chemical" hypothyroidism without clinical evidence of the disease was suggested. In murine hepatosplenic schistosomiasis the induction of hypothyroidism produced an exacerbation in the chronic stages of the disease.

Total serum thyroxine ( $T_4$ ) serum tri-iodothyronine uptake ( $T_3$ -uptake) and the free thyroxine index, were determined in seventeen hepatic schistosomiasis patients and ten controls by the competitive protein binding method. The mean values of  $T_4$ ,  $T_3$ -uptake and free thyroxine index showed no significant change between schistosomal patients and controls (Table 18). The mean values of liver function tests in schistosomal patients indicated impairment when compared with normal controls (Table 19). The lowest values of  $T_4$ ,  $T_3$ -uptake and free thyroxine index were found in patients with diminished serum albumin, raised thymol turbidity and zinc sulphate turbidity tests, but higher values of  $T_4$  and  $T_3$ -uptake were found in other patients with similar impairments of liver function tests. No correlation could be found between thyroid function tests and hypoalbuminemia, turbidity tests or serum bilirubin.

In man, dwarfism and infantilism are encountered in prepubertal cirrhosis, while various stages of disturbed

spermatogenesis and loss of libido occur if the disease occurs at a later stage in life.

Follicle-stimulating hormone (FSH) and luteinizing hormone (LH) were determined in twenty-nine adult male hepatic schistosomiasis patients and twenty controls by radioimmunoassay (Table 20). FSH and LH levels were significantly higher in hepatic schistosomiasis patients.

Urinary estrogens determined by chromatography and colorimetry in eight hepatic schistosomiasis patients and ten controls were  $46.86 \pm 9.05$  ug/24 hours and  $10.86 \pm 2.72$  ug/24 hours respectively, being significantly higher in hepatic schistosomiasis patients.

The urinary 17-ketosteroids excretion was significantly lower in schistosomal patients than in controls ( $4.95 \pm 3.30$  ug/24 hours and  $14.08 \pm 2.68$  ug/24 hours respectively).

The increased urinary estrogens excretion in hepatic schistosomiasis signifies high estrogen levels in the blood. Serum estrogens and androgens could not be determined because of unavailability of a liquid scintillation counter. The diminished urinary 17-ketosteroids excretion could be the result of direct inhibitory effect of elevated blood estrogens on androgens production or increased rate of androgen-estrogen biotransformation. Low androgen production increases FSH

secretion while estrogens decrease its level. The secretion of LH is also increased by low androgen levels but is less sensitive to inhibition by estrogens.

#### Haematological Studies:

Peripheral blood indices showed mild grade anaemia which is mostly hypochromic and normocytic, reticulocytic count was within the normal range, total leucocytic count within the normal range while differential leucocytic count showed eosinophilia.

Myelograms revealed hypercellular bone marrow, megakaryocytes were normal in number but showed moderate to poor platelet production, there was increase in the eosinophilic series, increased plasma cells, shift to the left of neutrophils and normoblastic reaction.

The red cell survival was determined using Cr-51 in nine patients with hepatosplenomegalic schistosomiasis. The Cr-51 red cell tagged half life varied between 15½ to 30 days with a mean of 23.1 days. Four patients were below the lower limit of normal of 25 days (15½, 16½, 18 and 20 days). The shortened red cell half-life in some cases pointed to the presence of a hemolytic element in the causation of anaemia of schistosomiasis.

Hepatic schistosomiasis is commonly accompanied by bleeding manifestations. Multiple factors interact in the pathogenesis of bleeding. Some of these factors were studied and included screening tests of blood coagulation (bleeding

time, whole blood clotting time, platelet count, prothrombin activity and activated partial thromboplastin time); platelet adhesion and platelet aggregation, fibrinogen estimation and euglobulin lysis time.

In hepatic schistosomiasis the prothrombin and activated partial thromboplastin times were significantly prolonged, a moderate degree of thrombocytopenia was demonstrated, platelet adhesiveness significantly decreased and platelet aggregation significantly delayed, fibrinogen estimation comparable to controls and fibrinolytic activity significantly accelerated (Table 24). The hepatic schistosomiasis patients were further divided to two groups, bleeders and non-bleeders and comparisons would be made.

Prolongation of prothrombin and activated partial thromboplastin times is attributed to defective hepatic synthetic function. Thrombocytopenia is due to diminished platelet production, maturation arrest and defective release. Shortening of the euglobulin lysis time reflects increased fibrinolytic activity and is attributed to impaired hepatic clearance of plasminogen activators.

Histochemical Studies:

Histochemical studies of wedge liver biopsies from twenty hepatic schistosomiasis patients included succinic dehydrogenase, alkaline phosphatase, acid phosphatase and non-specific esterase enzymatic activity. Reduced activity of succinic dehydrogenase all over the hepatocytes indicated diminished metabolic activity and diminished energy production while the increased enzymatic activity in Von Kupffer's cells indicated increased phagocytosis. The reactivity of alkaline phosphatase enzyme was diminished in the bilharzial liver. This might be due to degenerative changes in the liver. The acid phosphatase enzymatic activity was increased especially in Von Kupffer's cells indicating increased phagocytic power. The increased activity of non-specific esterase enzyme in hepatic schistosomiasis could indicate increased fatty change in the liver.

In these patients liver function tests were only slightly impaired yet marked abnormalities of enzymatic activities could be detected by histochemical studies.

Notes:

The original research project was suggested in 1972 and finally accepted in Juin, 1974. During this period the determination of plasma cortisol and urinary V.M.A. output in the basal state and after hypoglycemic and pyrogenic stimuli was performed and there was no need to repeat these tests.

In the sphere of carbohydrate metabolism, glucose tolerance and immunoreactive insulin determination was originally suggested. Both had been performed and in addition growth hormone levels in the basal state and after hypoglycemia were determined.

As regards blood lipids, not only free fatty acids were determined as originally suggested but also cholesterol, cholesterol ester, phospholipids, triglycerides and lipoproteins and the effect of portosystemic collaterals on their levels were studied. An intravenous fat tolerance test was also performed. Pl fractionation was also done as well as lipoprotein lipase activity.

Serum proteins were studied as originally suggested and in addition serum and urinary amino acids. An ammonia tolerance test was also performed.

The hematological studies included in addition to what was suggested studies of factors of coagulation and red cell survival.

We added also some endocrinological studies including thyroid hormones, serum follicle-stimulating and luteinizing hormones and urinary estrogens and 17-ketosteroids.

Papers Under Publication:

1. Intravenous fat tolerance in hepatic schistosomiasis.  
Annals of Tropical Medicine and Parasitology.
2. Histochemical and histopathological changes in hepatic schistosomiasis.  
The Alexandria Medical Journal.
3. Thyroid function in hepatic schistosomiasis.  
Annals of Tropical Medicine and Parasitology.
4. The effect of portosystemic collaterals on serum lipids in patients with schistosomal hepatic fibrosis.
5. Glucose tolerance, plasma insulin, growth hormone and free fatty acids in hepatic schistosomiasis.  
Annals of Tropical Medicine and Parasitology.

Papers Under Preparation:

1. Serum and urinary amino acids in hepatic schistosomiasis.
2. Coagulation factors in hepatosplenic bilharziasis.
3. Sex hormones in schistosomal hepatic fibrosis.
4. Ammonia tolerance in compensated hepatic schistosomiasis.
5. Red cell survival in hepatic schistosomiasis.
6. Phospholipids in schistosomal hepatic fibrosis.
7. Lipoprotein lipase activity in bilharzial hepatic fibrosis.

Table 1

Fasting Blood Sugar (mmol/l) and Glucose  
disappearance rate (percent per minute )

Mean  $\pm$  S.D.

	Controls (12)	Schistosomal (49)
Fasting Blood Sugar	4.16 $\pm$ 0.45	4.84 $\pm$ 0.79
Glucose disappearance rate	1.64 $\pm$ 0.26	1.27 $\pm$ 0.189

Table 2

The Blood Sugar percent Maximum drop after  
exogenous insulin administration, Mean $\pm$ S.D.

Controls (10)	52.60 $\pm$ 9.85
Schistosomal (13)	51.25 $\pm$ 11.33

Table 3  
Plasma IRI (nmol/l) during intravenous glucose tolerance tests,  
mean  $\pm$  S.D.

	F	4'	7'	10'	20'	40'	60'	90'	120'
Controls (9)	0.085 $\pm 0.038$	0.598 $\pm 0.298$	0.635 $\pm 0.327$	0.613 $\pm 0.319$	0.471 $\pm 0.239$	0.363 $\pm 0.272$	0.172 $\pm 0.128$	0.114 $\pm 0.054$	0.098 $\pm 0.055$
Schistosomal (18)	0.111 $\pm 0.04$	0.613 $\pm 0.506$	0.640 $\pm 0.518$	0.609 $\pm 0.605$	0.428 $\pm 0.350$	0.373 $\pm 0.400$	0.273 $\pm 0.198$	0.169 $\pm 0.101$	0.118 $\pm 0.058$
P	$> 0.05$	$> 0.05$	$> 0.05$	$> 0.05$	$> 0.05$	$> 0.05$	$> 0.05$	$< 0.05$	$> 0.05$

Table 4

HGH (nmol/l) During Intravenous Insulin Sensitivity Tests Mean  $\pm$  S.D.

	0	20'	30'	45'	60'	90'	120'
Controls (10)	0.015 $\pm$ 0.006	0.029 $\pm$ 0.027	0.112 $\pm$ 0.098	0.442 $\pm$ 0.290	0.547 $\pm$ 0.530	0.440 $\pm$ 0.451	0.270 $\pm$ 0.232
Schistosomal (13)	0.023 $\pm$ 0.022	0.095 $\pm$ 0.164	0.183 $\pm$ 0.190	0.480 $\pm$ 0.269	0.525 $\pm$ 0.252	0.423 $\pm$ 0.247	0.239 $\pm$ 0.111
P	$<0.05$	$<0.05$	$<0.05$	$<0.05$	$<0.05$	$<0.05$	$<0.05$

HGH = Human Growth Hormone.

Table 5

Fasting F.F.A. levels (umol/l) mean  $\pm$  S.D.

Controls (12)	209 $\pm$ 97
Schistosomal (49)	592 $\pm$ 173
P	< 0.05

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Table 6

Mean Serum Lipids (mg/100 ml) in Schistosomal Patients  
and Controls (mean  $\pm$  S.D.)

TL = total lipids. CH = total Cholesterol. CE = Cholestrol ester  
% Esterf. = % esterification of cholesterol. PL = Phospholipids-  
TG = Triglycerides Lip = alpha lipoprotein

Group	TL	CH	CE	%Esterf.	PL	TG	$\alpha$ -Lip
Bilh.	488	163	98.5	58.6	170	75	19.0
(41)	$\pm 13.5$	$\pm 30.4$	$\pm 18.3$	$\pm 4.4$	$\pm 23.2$	$\pm 23.2$	$\pm 4.2$
Control	620	205	142	69	214	90	27
(81)	$\pm 89.9$	$\pm 31.6$	$\pm 23.9$	$\pm 5.4$	$\pm 30.6$	$\pm 29.5$	$\pm 4.8$
P	$< 0.001$	$< 0.001$	$< 0.001$	$< 0.001$	$< 0.001$	$< 0.005$	$< 0.001$

Table 7

Mean Serum Lipids (mg/100 ml) in Schistosomal Patients  
with (group A) and without (group B) evidences of  
Portosystemic Collaterals (mean  $\pm$  S.D.)

Group	TL	CH	CE	%Esterf.	PL	TG	$\alpha$ -Lip
Group A	436.7	143.3	85.3	55.4	158	70.1	19.2
(18)	$\pm 50.3$	$\pm 20.7$	$\pm 11.8$	$\pm 10.9$	$\pm 20.4$	$\pm 16.5$	$\pm 3.9$
Group B	522.1	180.6	106.4	59.3	187.5	75.6	19.8
(14)	$\pm 58.7$	$\pm 19.3$	$\pm 13.4$	$\pm 3.7$	$\pm 17$	$\pm 25.8$	$\pm 5.4$
P	$< 0.001$	$< 0.001$	$< 0.001$	$> 0.05$	$< 0.001$	$> 0.05$	$> 0.05$

Table 8

Mean Serum Lipids (mg / 100 ml) in Schistosomal Patients  
(group A) with Evidences of Portosystemic Collaterals  
Before and After Operation (mean  $\pm$  S.D.)

Group	TL	CH	CE	%Esterf.	PL	TG	$\alpha$ -Lip
Before	414.3	139	74.1	53.6	153.6	65.7	17.4
(7)	$\pm 60.1$	$\pm 20.2$	$\pm 7.9$	$\pm 8$	$\pm 23.9$	$\pm 17.4$	$\pm 2.5$
After	530.9	183.1	104.5	56.4	190.0	77.0	17.1
(7)	$\pm 78.8$	$\pm 24.3$	$\pm 20.3$	$\pm 2.3$	$\pm 24.3$	$\pm 15.8$	$\pm 1.8$
P	$<0.01$	$<0.001$	$<0.01$	$>0.05$	$<0.01$	$>0.05$	$>0.05$

Table 9

Mean Serum Lipids (mg / 100 ml) in Schistosomal Patients  
(group B) with no Evidences of Portosystemic Collaterals  
Before and After Operation (mean  $\pm$  S.D.)

Group	TL	CH	CE	%Esterf.	PL	TG	$\alpha$ -Lip.
Before	537.8	183.5	113.8	61.7	189.3	78.5	22.8
(4)	$\pm 36.1$	$\pm 14$	$\pm 9.2$	$\pm 3.6$	$\pm 16.7$	$\pm 9.2$	$\pm 6.2$
After	547.3	187.8	111	61.5	193	80	20.8
(4)	$\pm 31.0$	$\pm 18.7$	$\pm 8.8$	$\pm 3.9$	$\pm 11.1$	$\pm 5.3$	$\pm 4.3$
P	$>0.05$	$>0.05$	$>0.05$	$>0.05$	$>0.05$	$>0.05$	$>0.05$

Table 10

Mean Serum Lipids (mg / 100 ml) in Schistosomal Patients  
with (group A) and without (group B) Evidences of Porto-  
systemic Collaterals after Operation (mean  $\pm$  S.D.)

Group	TL	CH	CE	%Esterf.	PL	TG	$\alpha$ -Lip
Group A	530.9	183.1	104.5	56.4	190.0	77.0	17.1
(8)	$\pm 78.8$	$\pm 24.3$	$\pm 20.3$	$\pm 2.3$	$\pm 24.3$	$\pm 15.6$	$\pm 1.8$
Group B	547.3	187.8	111	61.5	193.8	80.0	20.8
(4)	$\pm 31.0$	$\pm 18.7$	$\pm 8.8$	$\pm 3.9$	$\pm 11.1$	$\pm 5.3$	$\pm 4.3$
P	$> 0.05$	$> 0.05$	$> 0.05$	$> 0.05$	$> 0.05$	$> 0.05$	$> 0.05$

Table 11

TG Increment in mg per 100 ml after I.V. Intralipid (0.3g/kg)

Time (Minutes) After Intralipid	Controls		Schistosomal Patients		P
	Mean	$\pm$ S.D.	Mean	$\pm$ S.D.	
10	233	72.9	123	21.8	$< 0.01$
20	205	64.4	98	20.2	$< 0.01$
30	181	62.2	78	18.5	$< 0.01$
40	159	51.3	60	15.9	$< 0.01$
50	149	43.3	45	15.5	$< 0.01$
60	122	42.4	31	14.7	$< 0.01$
90	85	37.3	22	16.2	$< 0.01$
100	72	15.1	19	15.2	$< 0.01$

Table (12)

Plasma FFA (uEq/L) increment after I.V. Intralipid

Time (Min) After Intralipid	Controls		Schistosomal Patients		P
	Mean	$\pm$ S.D.	Mean	$\pm$ S.D.	
10	44	26.6	130.7	69.2	$< 0.01$
20	102	71.5	202	83.1	$< 0.01$
30	120	71.3	250	145.0	$< 0.01$
40	129	69.8	230	168.6	$< 0.05$
50	133	64.5	181	48.9	$< 0.05$
60	123	68.2	123	131.7	N.S.
90	76	41.3	24	38.4	$< 0.01$
120	56	36.9	-50	84.0	$< 0.01$

Table 13

Serum Proteins (Total and Electrophoretic Pattern)

	Total Serum Proteins G/L	Albumin G/L	$\alpha_1$ -Globulin G/L	$\alpha_2$ -Globulin G/L	$\beta$ -Globulin G/L	$\gamma$ -Globulin G/L
Controls (60)	68.5 + 6.4	41.0 + 4.2	2.4 + 0.37	5.0 + 0.94	6.98 + 1.9	15.7 + 3.3
Bilharzial (49)	78.4 + 3.5	32.6 + 6.0	2.6 + 0.3	7.3 + 1.3	7.7 + 1.5	28.1 + 6.5
P	< 0.0005	< 0.0005	< 0.0025	< 0.0005	< 0.025	< 0.0005

Table 14

Plasma Amino Acids Ug/ml in 23 Hepatic Schistosomiasis Patients  
(group 1) and 8 Controls (group 11) Mean  $\pm$  S.D.

	Shistosomal	Controls	P
Cystine & cysteine	25.6 $\pm$ 10.05	25.6 $\pm$ 8.46	> 0.05
Lysine	18.6 $\pm$ 5.80	21.2 $\pm$ 5.41	> 0.05
Histidine	12.7 $\pm$ 6.95	17.95 $\pm$ 5.39	> 0.05
Arginine	18.0 $\pm$ 7.33	20.7 $\pm$ 7.28	> 0.05
Glutamic acid	13.1 $\pm$ 5.80	18.28 $\pm$ 5.60	< 0.05
Serine	13.2 $\pm$ 5.48	12.8 $\pm$ 3.5	> 0.05
Aspartic acid	4.8 $\pm$ 2.78	5.31 $\pm$ 2.5	> 0.05
Glycine	17.81 $\pm$ 6.15	20.53 $\pm$ 8.10	> 0.05
Threonine	14.9 $\pm$ 3.89	15.67 $\pm$ 4.74	> 0.05
Glutamine	32.6 $\pm$ 12.71	50.26 $\pm$ 16.65	< 0.05
Alanine	31.57 $\pm$ 12.73	28.2 $\pm$ 10.15	> 0.05
Tyrosine	12.35 $\pm$ 4.52	12.97 $\pm$ 4.20	> 0.05
Methionine	9.23 $\pm$ 3.98	6.22 $\pm$ 1.92	< 0.05
Valine	11.98 $\pm$ 7.71	21.1 $\pm$ 6.16	< 0.05
Phenylalanine	11.11 $\pm$ 3.95	12.1 $\pm$ 5.42	> 0.05
Leucine & isoleucine	29.38 $\pm$ 16.50	32.8 $\pm$ 11.10	> 0.05
Tryptophane	5.46 $\pm$ 6.29	5.46 $\pm$ 5.49	> 0.05

Table 15

Urinary Amino Acids (mg/24 hours) in Hepatic Schistosomiasis  
Patients (group I) and Controls (group II)-Mean±S.D.

	Shistosomal	Controls	P
Cystine & cysteine	133.58 ± 78.6	15.41 ± 3.44	< 0.05
Lysine	25.12 ± 16.68	9.59 ± 2.96	< 0.05
Histidine	86.03 ± 34.46	94.13 ± 21.13	> 0.05
Arginine	10.81 ± 10.16	5.32 ± 1.31	> 0.05
Glutamic acid	5.18 ± 2.83	5.44 ± 1.68	> 0.05
Serine	40.48 ± 8.12	38.93 ± 6.85	> 0.05
Aspartic acid	5.36 ± 2.85	3.30 ± 1.08	> 0.05
Glycine	79.86 ± 50.81	96.34 ± 21.49	> 0.05
Threonine	27.17 ± 11.56	13.37 ± 4.91	< 0.05
Glutamine	75.02 ± 40.52	62.72 ± 11.41	> 0.05
Alanine	24.94 ± 12.59	32.81 ± 6.49	> 0.05
Tyrosine	44.6 ± 21.7	22.00 ± 5.70	< 0.05
Methionine	16.84 ± 7.85	4.86 ± 1.49	< 0.05
Valine	14.24 ± 6.21	8.62 ± 2.77	> 0.05
Phenylalanine	24.75 ± 8.18	13.64 ± 4.65	< 0.05
Leucine & isoleucine	32.24 ± 12.28	42.06 ± 15.59	> 0.05
Tryptophane	3.26 ± 3.01	0.80 ± 0.87	< 0.05

Table 16

Ammonia Tolerance in Hepatosplenic Schistosomiasis  
(Fasting and Increment Values, Mean  $\pm$  S.D.)

	Fasting	30'	60'	90'	120'
Group A.	153.84	22.47	23.85	17.24	- 2.51
	$\pm$ 44.59	$\pm$ 15.81	$\pm$ 13.29	$\pm$ 15.94	$\pm$ 10.53
Group B.	127.5	125.39	109.17	80.99	51.96
	$\pm$ 32.42	$\pm$ 43.42	$\pm$ 61.64	$\pm$ 25.18	$\pm$ 41.49
P	$< 0.05$	$< 0.0025$	$< 0.0125$	$< 0.0005$	$< 0.0125$

Table 17

Liver Function Tests in Patients with Mild (Group A) and Severe (Group B) Impairment of Ammonia Tolerance (Mean and  $\pm$ S.D.)

	Serum Bilirubin mg%	Alkaline Phosphatase K.A.	Thymol Turbidity units	Zinc $\text{SO}_4$ Turbidity units	Serum Albumin g/L	Gamma Globulin g/L	SgOT units	SgPT units
Ep A	0.6 $\pm$ 0.3	16.8 $\pm$ 7.26	5 $\pm$ 0.5	11 $\pm$ 3.5	37.8 $\pm$ 6.8	22.3 $\pm$ 6.4	25.5 $\pm$ 5.2	28 $\pm$ 8.4
Ep B	0.7 $\pm$ 0.28	20 $\pm$ 14.2	6 $\pm$ 2.16	12.5 $\pm$ 7.9	35.7 $\pm$ 5.7	23.8 $\pm$ 7.4	52.5 $\pm$ 12.3	61.5 $\pm$ 32.4
P	$>0.05$	$>0.05$	$>0.05$	$>0.05$	$>0.05$	$>0.05$	$<0.01$	$<0.05$

Table 18

 $T_4, T_3$ -uptake and free thyroxine index (Mean  $\pm$  S.D.)

	No. of cases	$T_4$ -uptake nmol/l	$T_3$ -uptake percent	Free thyroxine index
Controls	10	120.8 $\pm$ 20.6	110.37 $\pm$ 5.88	8.5 $\pm$ 1.38
Schistosomal	17	110.16 $\pm$ 25.6	112.39 $\pm$ 7.55	7.63 $\pm$ 1.73

Table 19

## Liver Function Tests (Mean and Range)

	Serum Total Bilirubin umol/l	Alkaline Phosphatase IU/l	Aspartate Aminotransferase IU/l	Alanine Aminotransferase IU/l	Rhytol Turbidity units	Zn SO <sub>4</sub> Turbidity units
Controls (10)						
Mean	6.67	42.4	8.94	7.69	2.25	4.53
Range	3.42-13.68	21.3-92.3	2.4-19.2	2.4-16.8	0-4	2-8
Schistosomal (17)						
Mean	12.66	106.5	15.84	16.80	6.3	10.7
Range	5.13-27.36	56.8-124.6	4.8-33.6	9.6-28.8	2-12	4-20
P	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05

TABLE. 20  
FSH and LH (ng/ml) in hepatic Schistosomiasis  
Patients and Controls, Mean  $\pm$  S.D.

	FSH	LH
Controls (20)	1.47 $\pm$ 1.22	2.26 $\pm$ 1.07
Schistosomal (29)	2.88 $\pm$ 2.45	3.42 $\pm$ 2.71
P	0.01	0.025

FSH. = Follicle stimulating hormone.

LH = Luteinizing hormone.

Table 21

## Haemostasis in Bilharzial Patients and Controls

	Bilharzial	Controls	P
Bleeding time			
min-sec	3'33"± 50"	3'26"±35"	> 0.05
Clotting time			
min sec	6'37"±1'50"	4'42"±49"	< 0.0005
Prothrombin activity			
percent	65.05± 8.64	100 ± 4.39	< 0.0005
Kaolin-cephalin			
seconds	65.15±10.4	49.20±22.76	< 0.0005
Platelets			
thousands/cmm	217.4 ±72.69	287.86±49.63	< 0.0005
Platelet adhesiveness			
percent	19.93± 4.92	28.8 ± 5.3	< 0.0005
Platelet aggregation			
seconds	29.88± 7.51	22.9 ± 3.75	< 0.0005
Fibrinogen			
mg %	232.8 ±40.7	334.53±55.88	> 0.05
Fibrinolytic activity			
Hours-minutes	3h8'± 56'	4h25'± 27'	< 0.0005